24 HOUR URINE SODIUM EXCRETION IN NEWLY DIAGNOSED HYPERTENSIVE PATIENTS

Dissertation submitted to

THE TAMILNADU

DR.M.G.R.MEDICAL UNIVERSITY

In partial fulfillment of the regulations

For the award of the degree of

M.D.BRANCH – I

GENERAL MEDICINE

GOVT.STANLEY MEDICAL COLLEGE &HOSPITAL

THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY

CHENNAI, TAMILNADU.

INDIA

MARCH -2010

CERTIFICATE

This is to certify that this dissertation entitled "**24 HOUR URINE SODIUM EXCRETION IN NEWLY DIAGNOSED HYPERTENSIVE PATIENTS**" submitted by Dr.G.ANANDH to The TamilNadu Dr. M.G.R. Medical University, Chennai is in partial fulfillment of the regulations for the award of M.D DEGREE BRANCH I (General medicine) and is a bonafide research work carried out by him under direct supervision and guidance.

Signature of the unit chief

Signature of the HOD

Signature of the Dean

DECLARATION

I solemnly declare that the dissertation titled **"24 HOUR URINE SODIUM EXCRETION IN NEWLY DIAGNOSED HYPERTENSIVE PATIENTS"** was done by me at Government Stanley Medical College and Hospital during 2008-2009 under the guidance and supervision of **Prof.Dr.A.GOWRISHANKAR M.D**.

This dissertation is submitted to the **TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY** towards the partial fulfillment of regulations for the award of M.D. Degree. (BRANCH – I) in **GENERAL MEDICINE.**

Place: Chennai.

Date: DR.G.ANANDH.

ACKNOWLEDGEMENT

At the outset I wish to thank our Dean, Govt.Stanley Medical College and Hospital , **DR.A.PRIYA M.S.D.O** .,for permitting me to carry out this study in our hospital.

I express my sincere thanks to **Prof.DR.S.RAMASAMY, M.D.,** Professor and Head of Department of General medicine for his guidance in conducting the study.

I sincerely thank my professor and unit chief **Prof.DR.A.GOWRISHANKAR, M.D**., for his eminent and continuous guidance in the study.

I am also thankful to my Assistant professors in Department of medicine, **DR.NALINI KUMARAVELU, M.D.,** and **DR.S.CHANDRASEKAR, M.D.,** and **DR.G.VASUMATHI, M.D.,** Registrar, for their constant encouragement, timely help and critical suggestions.

I thank my co-postgraduates for helping me in this study.

Last but not the least I sincerely thank all those patients and controls who participated in this study, for their co-operation.

CONTENTS

INTRODUCTION

Hypertension is one of the leading causes of death and disability among adults all over the world. It remains the major risk factor for coronary, cerebral and peripheral vascular disease. Essential hypertension comprises more than 90% of hypertension[1].

Hypertension is an emerging health problem in India. when majority of people come to know they have hypertension , they have already advanced in to stage with target organ damage – fatal stroke or myocardial infarction or irreversible renal damage unfortunately even in developed countries like U.S.A. fifty million people are found to have hypertension. Of these 70% are aware of their diagnosis but only 50% are receiving only treatment and only 25% are under control[2].

In addition to a primary increase in cardiac function propelled by overactive sympathetic nervous system, primary retention of salt and water by kidney, other factors contributing to hypertension are hereditary predisposition and high sodium and low potassium intake and excretion. Positive correlation exists with high sodium intake and increase in blood pressure.

Recent population based studies have shown a positive association between salt excretion and blood pressure, however some studies have negated this association[3]. Blood pressure was directly related to sodium intake and inversely and independently related to potassium intake.[3]

A recent study by Mohan et al[4] reports a prevalence of 20% in Chennai in the CURES study. Rural areas also showed a similar increase in prevalence of hypertension (~5-10%) although the rise was not as steep as in urban areas. Our present study was aimed at studying relationship of sodium and potassium excretion, and body mass index with blood pressure in normotensive and hypertensive people.

AIMS AND OBJECTIVES

1. To study 24 hour urine sodium excretion in newly diagnosed hypertensive patients.

2. To correlate 24 hour urine sodium excretion with respect to their stages of hypertension as in Joint National Committee JNC VII and with respect to their body mass index.

REVIEW OF LITERATURE

ESSENTIAL HYPERTENSION

An elevated arterial pressure is one of the most important public health problem and despite widely recognised high prevalence and associated danger , it remains moderately treated in majority of patients .Its common ,readily detectable and usually easily treatable and if left untreated can lead to serious morbidity and mortality from cardiac, cerebrovascular, vascular and renal disease. Adequate hypertension control remains elusive because of the asymptomatic nature of disease for first 15-20 years even as it progressively damage the cardiovascular system[5].Although our understanding of the pathophysiology of hypertension has increased, in 90% to 95% of cases etiology is still unknown.

Definition and classification

Blood pressure is distributed in atypical bell shaped curve within overall population. As seen in multiple risk factor intervention trial (MRFIT), the long term risk for cardiovascular mortality rise progressively over the entire range of blood pressure with no threshold that clearly identifies the potential danger. Therefore the definition of hypertension is somewhat arbitrary and usually taken as that level of pressure associated with doubling of long term risks.

TABLE 1

JNC-7 CLASSIFICATION OF BLOOD PRESSURE FOR ADULTS ≥ 18 YEARS.*

SI.NO	CATEGORY	SYSTOLIC B.P (mm of Hg)		DIASTOLIC B.P (mm of Hg)
1	Normal	<120	and	<80
2	Prehypertension	120-139	or	80-89
3	Hypertension Stage 1	140-159	or	90-109
	Stage 2	≥160	or	≥110

*[Source, JAMA 2003; 289:2560]

Prevalence

Cardiovascular diseases account for a large population of all deaths and disability worldwide. In 1990 there were 5.2 million deaths from cardiovascular diseases.

Hypertension is directly responsible for 57% of all stroke deaths and 24% of all coronary heart disease deaths. This fact is important because

hypertension is a controllable disease and a 2mm Hg population-wide decrease prevent 151,000 strokes and 153,000 coronary heart disease deaths in india.[6, 7]

The average blood pressure levels in china were systolic: 118±18mm Hg and diastolic: 76.6 ± 11mmHg in a sample of 10,076 urban and rural subjects 35-54 years of age. In India Gupta et al reported mean systolic blood pressure(men :rural 127±14 urban, 125±17;women:rural 124±13,urban 126±18)and diastolic blood pressure (men; rural 81±8 urban 81±9;women rural 80±8 urban 81±12) levels in western Indian urban and rural subjects aged ≥20years[8].Systolic blood pressure has increased in Indian men aged 40-49 years from 123±11mmHg in 1959 to 128±17mmHg in 1995.[9]

Recent studies among Indians have shown a high prevalence of hypertension in both rural and urban areas.[10,11] The prevalence rate of hypertension in India done by various study group is shown in Table 2 given in next page.

TABLE 2

INDIAN HYPERTENSION PREVALENCE STUDIES (B.P >140mmHg.)

First author	Age Group	Place	Sample size		Prevalence (%)	
			men	women	Men	Women
Gupta R 1995	20-75	Jaipur	1415	797	29.5	33.5
Joseph A 2000	20-89	Trivandrum	76	130	31	41.2
Anand MP 2000	30-60	Mumbai	1521	141	34.1*	-
Mohan V 2000	20-70	Chennai	518	657	14*	-
Gupta R 2002	20-75	Jaipur	550	573	36.4	37.5
Gupta PC 2004	18-60	Mumbai	40067	59522	43.8	44.5

*Gender specific data not available.

*Source J human hypertension 2004; 18:73-78

Awareness status of hypertension in India is poor. In urban population of Mumbai there was very low awareness of hypertension and only 6.1%males and 10.1% females were aware of hypertension in early 1990[12].In Jaipur also it has been reported that 11% of male and 16% female hypertensives were aware of their condition[13].Higher awareness of hypertension has been reported among more educated population in Kerala and among parsis in Mumbai[14,15]. In community dwelling individuals in Kerala (n=357, mean age70 yrs) hypertension was present in 51.8%and 44.9% of the individuals were aware of their condition[14].Bharucha and Kuruvilla[15] reported that 53% of men and 44%were aware of their hypertensive status, although 905 have their blood pressure measurement in past. This level of awareness is similar to reported from many developed countries and shows that within India there is wide variation in hypertensive awareness status.

Natural history and complication

The pathological hallmark of untreated hypertension is acceleration of atherosclerosis. The higher the blood pressure, the more likely cardiovascular diseases will develop prematurely. If untreated 50% of hypertensive patient die of coronary artery disease and congestive heart failure, about 33%of stroke 10-15% of renal failure. A Meta analysis of nine major prospective studies shows a direct continuous and apparently independent

association of diastolic blood pressure with both coronary artery disease and stroke.[16]

In general vascular complications of hypertension can be considered as either hypertensive or atherosclerotic.

I. H**ypertensive complication**.

1. Accelerated malignant phase.
2. Hemorrhagic stroke.
3. Congestive heart failure.
4. Nephrosclerosis.
5. Aortic dissection.

II. **Atherosclerotic complication.**

1. Coronary artery disease.
2. Sudden death
3. Arrhythmias.
4. Atherothrombotic stroke.
5. Peripheral vascular stroke.

Overall cardiovascular risks

The degree of risks from hypertension can be categorized with reasonable accuracy by taking into account,

1. The level of blood pressure.
2. The biological nature of hypertension based on target organ damage.
3. The co-existence of other cardiovascular risk factor [17].

The goal of anti-hypertensive therapy should not only be reduction of blood pressure but also treating other risk factors. The major cardiovascular risks factors indicated in JNC-7 report are

1) Hypertension.
2) Cigarette.
3) Obesity.
4) Physical activity.
5) Dyslipidemia.
6) Diabetes mellitus.
7) Microalbuminuria or estimated glomerular filteration rate<60 ml/ min.
8) Age (>55 for men,>65 for women).

9) Family history of premature cardiovascular disease (<55 for men,<65 for women).

Systolic hypertension and Pulse pressure

Systolic blood pressure rises in a linear fashion with age, whereas diastolic blood pressure increases until age of 50 then levels off and began to fall .Isolated systolic hypertension is more common in younger subjects while isolated diastolic hypertension is more common in elderly. The underlying pathology process is loss of arterial tissue, which means that the pressure was created by left ventricular contraction, can no longer be dammed by aorta and major vessels.

Systolic blood pressure is a better predictor of cardiovascular system risk and isolated systolic hypertension is now recognised to be independent risk factor of cardiovascular disease .A wide pulse pressure has a similar influence on prognosis[18].

Gender differences

Hypertension is an important risk factor for cardiovascular disease in women. Although premenopausal women have lower blood pressure than age matched men, the prevalence of hypertension is higher in women than men after age of 65.Obesity is more common in middle aged and older women and is likely to contribute to cross over in prevalence. Oral contraceptive pills

increases risk of hypertension in younger women. The ratio of hypertension frequency in women versus men increases from 0.6 to 0.7 at age 30 to 1.1 to 1.2 at the age of 65 yrs.

Hypertension in Black population

Hypertension is common in black than in white and more common in urban than in rural blacks. Black individuals have a higher incidence of salt sensitive hypertension than white individuals and retain more sodium leading to expanded plasma volumes and lower plasma rennin activity. The complication of hypertension tends to be different in blacks with a higher incidence of left ventricular hypertrophy, stroke, renal failure and lower risk of coronary artery disease and is due to severity of hypertension in blacks and lower risk of coronary artery disease than white is due to more favourable lipid profiles.

MECHANISM OF PRIMARY HYPERTENSION

No single specific cause is referred to as primary in preference to essential. Blood pressure is product of cardiac output and peripheral vascular resistance(B.P=C.O*PVR).since persistent hypertension can develop only in response to an increase in cardiac output or a rise in peripheral resistance ,defects may be present in one or more of the multiple factors that affect these two forces. This can be described under the following headings

1. Non renal factors

2. Renal factors.

1) NON RENAL FACTORS

Primary hypertension is a complex multifactorial and polygenic disorder that results from an interaction between an individual's genetic background and various environmental factors.

Genetic predisposition

In studies of twins and family members in which degree of familial aggregation of blood pressure level is compared with the closeness of genetic sharing, the genetic contributions have been estimated to range from 30% to 60% [19]. Harrap suggested that the average population blood pressure is determined by the environment but the blood pressure rank within the distribution is decided largely by genes.[20]

Epidemiological data suggest that for population variability in blood pressure genetic factors contribute 30-35%, common household environment about 10-15% and non-familial factors for the remaining 50-55%.[21]

If genetic markers of a predisposition for the development of hypertension are found, then specific environmental manipulations could then be directed toward the susceptible subject[22].Pratt, from

his observations of bimodal distributions of blood pressure in some families with hypertensive subjects, proposed autosomal dominant mode of inheritance. Pickering proposed that blood pressure is a quantitative trait with genetic contribution which is polygenic.

Genome wide scanning strategy in sib-pairs has identified chromosomal regions on chromosomes 6, 5, 12, 15 which showed significant linkage to genes that influences inter individual blood pressure variation. There are several candidate genes within the identified group[23].

Polymorphism of genes involving RAS system, aldosterone synthesis and adrenergic receptors has been noted to be more common in the hypertensive than normotensive patients.[24] Genetic abnormalities may be monogenic in some rare forms of hypertension like glucocorticoid remediable aldosteronism, Liddle syndrome, and apparent mineralocorticoid excess.[25]

Fetal environment

Low birth weight, as a consequence of fetal under nutrition is followed by an increased incidence of high blood pressure later in life with an overall estimate that a 1kg lower birth weight is associated with a 2 to4mmHg higher systolic blood pressure in adulthood[26].Brenner & Chertow hypothesized that a decreased number of nephrons from intra uterine growth retardation could

very well serve as a permanent irreparable defect that eventuates in hypertension.[27]

Vascular remodelling

A number of factors increase peripheral resistance by both functional contraction and vascular remodelling and hypertrophy. Multiple vasoactive substances act as pressure-growth promoters resulting in both vascular contraction and hypertrophy simultaneously, but perpetuation of hypertension involves hypertrophy. Lever and Harrap[28] postulated that primary hypertension has two mechanisms similar to secondary hypertension a)a growth promoting process in children and b)a self perpetuating mechanism in adults.

Neurohumoral causes of primary hypertension

A large number of circulatory hormones and locally acting substances may be involved in the development of hypertension which causes hypertension by vascular hypertrophy, capillary rarefaction and unpaired microvascular dilation[29].

A. **Sympathetic nervous hyperactivity**

Young hypertensives tend to have increased levels of circulating catecholamines ,augmented sympathetic nerve traffic in muscles, faster heart rate and heightened vascular reactivity to alpha adrenergic agonists[30] .These changes could raise the blood pressure in a number of ways either alone

or in concert with stimulation of rennin release by catecholamines or by causing arteriolar and venous constriction or by increasing cardiac output or by altering the normal renal pressure-volume relationship.

B. Renin angiotensin system

Both as a direct pressor and as a growth promoter renin angiotensin system may be also involved in pathogenesis of hypertension. All functions of renin are mediated through the angiotensin II. This system is the primary stimulus for the secretion of aldosterone and hence mediates mineralocorticoid responses to varying sodium intake and volume overload when sodium intake is reduced or effective plasma volume shrinks the increase in renin- angiotensin II stimulates aldosterone secretion which in turn is responsible for a portion of the enhanced renal retention of sodium and water.

When large proportions of hypertensives are surveyed, only about 30 percent have low plasma renin activity levels, whereas 50 percent have normal levels and the remaining 20 percent have high levels [31]

Normal and high renin hypertension.

Some persons with primary hypertension have normal or high renin activity. The concept of “nephron heterogencity”[32] described by Sealy & collegues, assumes a mixture of normal and ischemic nephrons caused by afferent arteriolar narrowing. Excess renin from the ischemic nephrons could raise the

total blood renin levels to varying degrees and cause high renin levels in patients with primary hypertension.

C. Hyperinsulinemia/ Insulin resistance

An association between hypertension and hyperinsulinemia has been recognized for many years, particularly with accompanying obesity but also in about 20% of non-obese hypertensive patients[33].The hyperinsulinemia of hypertension arises as a consequence of resistance to effects of insulin on peripheral glucose utilisation. This association does not apply to pima Indians but it has been found in blacks, Asians and as well as whites. The impairment of the peripheral actions of the insulin results from a defect in the usual vasodilatory effect of insulin mediated through increased synthesis of nitric oxide, which normally counters the multiple pressors effects of insulin[34].

These pressor effects include activation of sympathetic activity, a trophic action on vascular hypertrophy, and increased renal sodium reabsorption resulting in a rise in blood pressure that may be either a primary cause of hypertension or, atleast, a secondary potentiator.

D. Endothelial cell dysfunction

The endothelium is now known to be the source of multiple relaxing and contracting substances of which niric oxide is an important vasodilator[35].The impairement of normal vasodilation in the insulin resistance syndrome has been

shown to involve failure to synthesize the normal endothelium derived relaxing factor (NO).

Nitric oxide

Hypertensive patients have been shown to have a reduced vasodilatory response to various stimuli of nitric oxide release that appears to be independent of the etiology of the hypertension and the degree of the gross vascular structural alteration. Impaired nitric oxide mediated vasodilation may promote abnormal vascular remodelling and may be involved in the greater propensity for vascular damage in blacks than whites.

Endothelin

Endothelin-1 causes pronounced and prolonged vasoconstriction and because blockade of its receptors improves endothelium dependent vasodilation in hypertensives patients.[36]

E) Minerals

Excess of lead and changing ratios among dietary sodium, potassium, calcium and magnesium have also been postulated in the pathogenesis of primary hypertension.[37]

2) RENAL RETENTION OF EXCESS DIETARY SODIUM.

A considerable amount of circumstantial evidence supports a role for sodium in the genesis of hypertension. To induce hypertension some of the excess sodium must be retained by the kidneys .such retention could arise in a number of ways.

1. Nephron heterogeneity, described as the presence of a sub population of nephrons that is ischemic either from afferent arteriolar vasoconstriction or from an intrinsic narrowing of lumen. Renin secretion from this subgroup of nephrons is tonically elevated. This increased renin secretion then interferes with capacity of normal nephrons to excrete sodium.

2. A resetting of the normal pressure natriuresis relationship- Guyton hypothesis.[38]

3. An acquired inhibitor of the sodium pump or other abnormalities in sodium pump or other abnormalities in sodium transport.[39]

4. Defensive responsiveness to atrial natriuretic hormone.[40]

ASSOCIATION OF HYPERTENSION WITH OTHER CONDITIONS

1) PHYSICAL ACTIVITY

Physical fitness may help prevent hypertension and persons who are already hypertensive may lower their blood pressure by regular

isotonic exercise. The relationship may involve insulin resistance because an increased resistance was coupled with low physical fitness in normotensive men with family history of hypertension.

2) ALCOHOL

Alcohol in larger amounts (more than 2 portions a day and more so when drunk in binges) increases blood pressure and arterial stiffness. The pressure effect of larger amount of alcohol reflects an increase in cardiac output and heart rate, possibly a consequence of increased sympathetic nerve activity. Alcohol also alters cell membranes and allows more calcium to enter, perhaps by inhibition of sodium transport.

Alcohol in small amounts (less than 1or 2 usual portions a day provides protection from coronary disease, congestive heart failure, stroke and dementia[41].And atleast in women, reduces incidence of hypertension[42].The reduction in coronary disease in persons who ingest small amount of alcohol reflect an improvement in lipid profile, a reduction in factors that encourage thrombosis, and a improvement in insulin sensitivity.

Framingham study showed small overall correlation between alcohol intake and blood pressure, but prevalence of hypertension was about 2 tiers higher among persons drinking 60 ounce

or more of ethyl alcohol per month than among those drinking less than 30 ounce per month.[43]

3)SMOKING

Cigarette smoking raises blood pressure, probably through nicotine induced release of nor- epinephrine from adrenergic nerve endings. In addition, smoking causes an acute and marked reduction in radial artery compliance independant of the increase in blood pressure. When smokers quit,a rise in blood pressure may occur, probably reflecting again on weight. Numerous studies have shown that smokers are thinner than non smokers and that smoking reduces weight. However they will have larger waist hip ratio than non smokers[44].

4) HEMATOLOGICAL FINDINGS.

Higher hematocrit are found in hypertensive persons and are associated with abnormal left ventricular filling on echocardiography[45].whole blood viscosity is increased by about 10% in persons with untreated mild hypertension, comparable to the increase in their peripheral resistance[46].Pseudo or stress polycythemia with high hematocrit and increased blood viscosity but contracted plasma volume, as well as normal red cell mass and serum erythropoietin levels are found in hypertension. High white blood cells count is predictive of hypertension.[47]

5) HYPERURICEMIA

Hyperuricemia is present in 25% individuals with untreated hypertension and in more than 75% of patients with malignant hypertension which are about 5 times the frequency found in normotensive persons[48].Hyperuricemia probably reflects decreased renal blood flow, presumably a reflection of nephrosclerosis.

6) **SLEEP APNOEA.**

Snoring and sleep apnoea are often associated with hypertension, which may in turn be induced by the increased sympathetic activity and endothelium release in response to hypoxemia during apnoea.[49]

7) HYPERCHOLESTROLEMIA

Hypercholesterolemia frequently co-exists with hypertension, at least in part because it impairs endothelium- dependent vasodilatation. Lipid lowering therapy restores the bio-availability of nitric-oxide, reduces arterial stiffness, and lowers blood pressure.[50]

8) OBESITY

The majority of patients with high blood pressure are overweight and hypertension is about 6 times more common than its in lean subjects.[51]

A 10 kg higher body weight is associated with 3mm Hg higher systolic and 2.3mm Hg higher diastolic blood pressure. These increases translate into an estimated 12% increase in the risk of coronary artery disease and 2.24% increase in the risk of stroke.

Body mass index in widely used as a correlation with excess body fat or adiposity but it does not convey information on required fat distribution. Body fat distribution plays a role as a risk factor for hypertension. An increase in waist hip ratio(WHR)>0.95 in male and 0.8 in female is an independent risk factor for the development of hypertension and is independently associated with hypertriglyceride and increased apoprotein.The waist circumference may be the better indicator of visceral fat than waist hip ratio.

HIP CIRCUMFERENCE- it is measured at the point of one third of the distance between the anterior superior iliac spine and patella.

WAIST CIRCUMFERENCE-it is measured half way between superior iliac crest and rib cage in mid-axillary line. It correlates well with systolic blood pressure and diabetes mellitus.

But to prevent wide gender differences we can use body mass index to compare obesity and hypertension.

BODY MASS INDEX=WEIGHT IN KG/HEIGHT IN m^2

TABLE-III

Proposed Classification of Body Mass Index In Adult Asians[52]

Classification	BMI (kg/m^2)
Underweight	<18.5
Normal range	18.5-22.9
Overweight	23
At risk	23-24.9
Obese I	25-29
Obese II	>30

* Source Am J Clin Nutr, 2000; 72:1067-1068

Metabolic Syndrome

According to ATP III guidelines metabolic syndrome is defined as a cluster of cardiovascular risks in a sliver individual –normally hypertension, diabetes mellitus, and abdominal obesity, low HDL increased TGL, procoagulant tendency and increased small LDL.

Obese individuals with high waist hip ratio have high incidence of hypertension than do with low waist hip ratio[53]. Visceral obesity is a striking risk factor for hypertension although body mass index is a strong determinant of

blood pressure, a visceral distribution of fat has an even greater relationship with development of hypertension[54].

Sodium metabolism and Sodium balance

Major intracellular particles are sodium and its accompanying anion chloride and bicarbonate, whereas potassium and organic phosphate esters (ATP, creatine phosphate and phospholipids) are the predominant intracellular fluid osmoles. Since sodium is largely restricted to the extracellular compartment, total body sodium content is a reflection of the extracellular fluid volume.

Sodium is actively pumped out of the cells by the $NA^{+}K^{+}$ATP ase pump. As a result 85 to 95 % of all sodium is extracellular and extracellular fluid volume is a reflection of total body sodium content .Changes in sodium concentration generally reflects disturbed water homeostasis, where as alteration in sodium content are manifested as extracellular fluid volume contraction or expansion and imply abnormal sodium balance.

Sodium intake

An individual eating a typical western diet consumes approximately 150 m mol of sodium chloride daily this normally exceeds basal requirements therefore dietary intake of sodium results in extracellular fluid volume

expansion which in turn promotes enhanced renal sodium excretion to maintain steady state sodium balance.

Sodium excretion

The regulation of sodium excretion is multifactorial and is the major determinant of sodium balance. Tubule sodium absorption is the major regulatory mechanism controlling sodium excretion. Almost two thirds of filtered sodium is reabsorbed in proximal convoluted tubule. Further reabsorption (35-30%) occurs in the thick ascending limb of loop of henle via apical Na^+ K^+3Cl^- co transporter. Distal convoluted tubule reabsorption of sodium(5%)is mediated by thiazides sensitive sodium chloride cotransporter.Final sodium reabsorption occurs in the cortical and medullary collecting ducts, the amount excreted being reasonably equal to the amount ingested per day.

SODIUM AND HYPERTENSION

1. Sympathetic Nervous System and Sodium Handling

The renal sympathetic nervous system directly stimulates the sodium reabsorption and renin release from the juxta glomerular apparatus. Several studies have linked sympathetic nervous system hyperactivity with greater than normal increase in blood pressure in response to a given sodium load[55]. Most authorities believe that mechanism by which the kidney causes hypertension is

impairment in excretion of sodium[56, 57]. This impairment may be attributed to genetic changes in various sodium exchangers in the proximal and distal tubules resulting in altered response to stimulation by the sympathetic nervous system and renin angiotensin –aldosterone system.

Interventional studies with sodium restriction and loading have revealed that the blood pressure responses in many hypertensives patients are salt sensitive .Salt loading of patients with essential hypertension results in an increase in blood pressure and in a net body sodium accumulation.

2.An increase in cytosolic free sodium concentration in cells of hypertensives compared with age and sex matched normotensive controls have been documented[58] .This results from altered activity of the $Na^{+}k^{+}$ antiporter and the Na^{+} Li^{+} counter transporter the increase in intracellular sodium is highly correlated with presence of an elevated diastolic blood pressure.

3. Most patients with essential hypertension have a defect in the pressure natriuresis curve, in which higher systemic pressures are required to excrete a sodium load.[59]

4. Another mechanism for decreased sodium excretion in patients with essential hypertension is enhancement of tubuloglomerular feedback.[58]

5. Alterations in intrarenal vasoactive mediators may be involved in the impairment of sodium excretion in patients with essential hypertension. There

may be low levels of renal vasodilators such as prostaglandins, dopamine and nitric oxide as well as elevated levels of renal vasoconstrictors such as angiotensin II and adenosine and increased activity of the renal sympathetic nervous system .Alterations in the levels of these agents could contribute to net sodium reabsorption because of their direct effects on tubular sodium transport.

Potassium balance

Potassium is the major intracellular cation. The normal plasma potassium concentration is 3.5 to 5 mmol /l whereas that inside the cell is about 150 mmol / l .therefore the amount of potassium in the extracellular fluid is less than 2%of total body potassium content. This is due to the principal result of the resting membrane potential and is crucial for normal neuromuscular function the basolateral $NA^{+}K^{+}$ATPase pump actively transports potassium in and out of the cell in a 2:3 ratio and the passively outward diffusion of potassium is quantitatively the most important factor that generates the resting membrane potential.

Potassium intake

The intake of individuals on an average western diet is 40 to 120 mmol/day.90% of which is absorbed by the gastrointestinal tract .immediately following meal most of the absorbed potassium enters cells as a result of the

initial elevation in the plasma potassium concentration and facilitated by the insulin release and basal catecholamine levels.

Potassium excretion

Renal excretion is the major route of elimination of dietary and other sources of excess potassium 90% of the filtered potassium is reabsorbed by the proximal convoluted tubule and the loop of henle. Proximally potassium is reabsorbed passively with sodium and water, whereas the luminal Na+K+2CL- co transporter mediates potassium uptake in the thick ascending limb of loop of henle, therefore potassium delivery to the distal nephron approximates dietary intake. The cell responsible for potassium secretion in the late distal convoluted tubule and cortical collecting duct is the principal cell.

MATERIALS AND METHODS

SETTING : This study was carried out in Hypertension outpatient department and wards of Department of General Medicine, Government Stanley medical college, Chennai.

STUDY DESIGN: Analytical study.

SAMPLE POPULATION: 100 CASES, 70 cases + 30 controls

PERIOD OF STUDY: October 2008 to October 2009.

ETHICAL COMMITTEE APPROVAL: The present study was approved by the ethical committee.

MATERIAL AND METHODS

Seventy newly diagnosed hypertensive patients as per criteria of JNC VII report. Controls were selected from the attendants accompanying cases except siblings.

Inclusion criteria

1. Newly diagnosed hypertension patients not started on treatment.
2. Hypertension as per criteria of JNC VII report i.e. blood Pressure 140/90 mmHg least at three different occasions.
3. Patients age above 18 years.
4. Both sexes were included.

Exclusion criteria

1. Patients of secondary hypertension
2. Patients on non-steroidal anti-inflammatory agents, diuretics.
3. Patients with congestive cardiac failure.
4. Patients with malignant hypertension.
5. Females on oral contraceptive medication.

6. Hypertensive patients on treatment

Control group

1. Same age and sex.

2. BP lower than the specified for hypertensive group

Consent

The study group thus identified was informed about the nature of the study and willing participants were included in the study after getting written informed consent from them.

Study subject and controls

Seventy newly diagnosed patients of hypertension from outpatient department and wards of Government Stanley medical college for the period of study, comprised the study group. Thirty controls were selected from the attendants of hypertensive patients living in the same environment other than their siblings. Incidentally in our study cases fall within the age group of 40 to 60 years, so controls were selected in the same age group.

Details of the study subjects

A detailed history of all subjects especially family history of hypertension, cardiovascular disease, renal disease, diabetes mellitus, history of

weight gain, sodium intake, alcohol use and symptoms suggestive of secondary hypertension. History of other cardiovascular risk factors (including obesity, smoking, hyperlipidemia, diabetes) and all prescribed and over the counter medications. All subjects were subjected to thorough physical examination which included all peripheral pulses including carotids to rule out atherosclerosis.

Examination of extremities for oedema, diminished and absent peripheral arterial pulsations, measurement of body mass index (BMI) calculated by weight/height2 (kg/m2),waist and hip circumferences were measured optic fundoscopic examination for any hypertensive changes, neck for distended veins, thyromegaly, cardiovascular, respiratory, abdominal and CNS examination.

Atleast three blood pressure measurements with same standard mercury sphygmomanometer and Littman's stethoscope in both supine and standing in both the arms were measured.

GUIDELINES FOR MEASURING BLOOD PRESSURE

I .CONDITIONS FOR THE PATIENT

A POSTURE

1.Patient should sit with back supported for about 5 minutes before taking a reading and the arm should be resting at heart height on a table, or other suitable property.

2. For diabetics and patients aged above 65 check postural changes by taking blood pressure immediately and two minutes after standing.

B. CIRCUMSTANCES

1. Do not move while taking reading.
2. Do not smoke for about 30 minutes before taking a reading. Do not consume any caffeine (coffee, soda, tea etc.) for about 30 minutes before taking a reading. Preferably do not to eat for at least 30 minutes before taking reading.
3. Have an empty bladder.
4. Home readings taken under various circumstances and 24 hr ambulatory recordings may be preferable.

II EQUIPMENT

A) CUFF SIZE:

The bladder should encircle and cover 2/3 of arm length. If not, place the bladder over the brachial artery, if bladder is small, high readings may result.

B) MANOMETER:

Sphygmomanometer is used for measuring the blood pressure.

3) TECHNIQUE

A) NUMBER OF READINGS.

a. Three readings are taken for each patient and the average of three readings is taken.

b. Initially, blood pressure is measured in both arms, if blood pressure differs, higher blood pressure reading is taken.

c. If arm pressure is elevated, blood pressure is measured in one leg, particularly in patients below age 30.

B) PERFORMANCE.

The bladder is inflated quickly to a pressure 20mmhg above the systolic, as recognized by disappearance of a radial pulse. The bladder is deflated 3mm Hg every second. The systolic blood pressure corresponds to appearance of korotkoff sounds and the diastolic blood pressure corresponds to the disappearance of korotkoff sounds(phase 5) .If korotkoff sounds are weak, have the patients raise the arm, open and close hand 5 to 10 times, after which bladder should be inflated quickly.

C)RECORDING

The blood pressure, patient position, the arm cuff size (eg.140/90,seated, right arm, large adult cuff,)are noted.

Urine albumin, sugar, microscopy and pH were done for all the subjects. A 12 lead ECG, ultrasonogram abdomen and chest x-ray were also taken.

Overnight (12hr) fasting blood sugar and urea was done by using diacetlyl monoxime (DAM) technique. Serum creatinine was estimated using COBAS auto analyser. Serum sodium and potassium was estimated using flame emission photometric method in an accurate manner.

TWENTY FOUR HOUR URINE STUDY

A single measurement of 24-h sodium excretion is a valid estimate of dietary sodium intake [61.] The 24-h urine collection was considered the most reliable method to evaluate sodium and potassium intake amounts [62, 63.]

All subjects were given thorough instructions about how to collect the 24hour urine specimen both orally and in written form. Sodium, potassium, protein and creatinine levels were estimated. Volume is accurately measured. The sample was analysed for accuracy with creatinine values. [Ref. Value 24 hrs Urine: MEN: 1040 - 2350 mg/24 Hrs WOMEN: 740 - 1570 mg/24 Hrs] and volume [>500 ml]. In our study all the samples collected were accurate. Hitachi 902 Multichannel analyser was used to measure electrolytes and Jaffe's method is used to measure creatinine.

Definitions used in present study

Essential hypertension

Hypertension was defined in accordance with JNC VII report as systolic blood pressure 140 mm hg and above and or diastolic blood pressure 90 mm hg

and above .The diagnosis of essential hypertension is based on the clinical examination and laboratory investigations . As the cases were newly diagnosed the duration of hypertension is not known.

Sodium and potassium normal values

The normal range for sodium was from 136 to 146 mmol /l .The normal range for serum potassium is from 3.5 to 5.1 mmol/l .The 24 hour urine sodium normal value is 40 - 220 mEq/24 hours and 24 hr urine potassium normal value is 25 - 125 mEq /24 hrs.

Obesity

According to proposed classification of weight by B.M.I in adult Asians, the patients with a BMI less than 18.5 were classified as underweight , 18.5 to 22.9 were classified as normal >23 were classified as overweight and >35 were classified as obese

Diabetes mellitus

Patients with fasting glucose > 126mg/dl or two hour plasma glucose >200 mg/dl or with symptoms of diabetes plus RBS>200 mg/dl were considered to be diabetic.

Left ventricular hypertrophy

Based on the Electrocardiographic findings which satisfy either the Sokolow lyon criteria or cornell voltage criteria. [64, 65]

Conflict of interest

There was no conflict of interest

Financial support

No.

Limitations

1. Arterial blood gas analysis was not done due to financial limitations.

2. Renal handling of sodium was not attempted as it was beyond the scope of the study.

3. Body water content was not associated with which may alter the level of sodium and potassium.

4. Tissue sodium and potassium was not measured.

5. Hormones related sodium and potassium handling in kidney was not measured

6. The salt intake of the patient could not be measured quantitatively and qualitatively because of social constraints.

7. A single measurement of 24-h sodium excretion is made because of social and financial constraints.

Statistical analysis

The collected data was entered in Microsoft excel spread sheet and analysed statistically. Student t values were applied for significance. Significance was considered if p value was below 0.05[95% confidence].

RESULTS AND OBSERVATIONS

The total number of subjects included in this study was 100.Among these 100 subjects 70 were cases and 30 were controls. Among cases 26 were stage I and 44 were stage II hypertension according to JNC VII.

Analysis of cases and controls with respect to age

The age of the subjects in the study group ranged from 40 to 60 years. The mean and standard deviation for cases and control were 53.7±4.88 and 51.9

± 4.89 respectively. The study group and the control group did not differ from each other statistically with reference to age. (Fig-1)

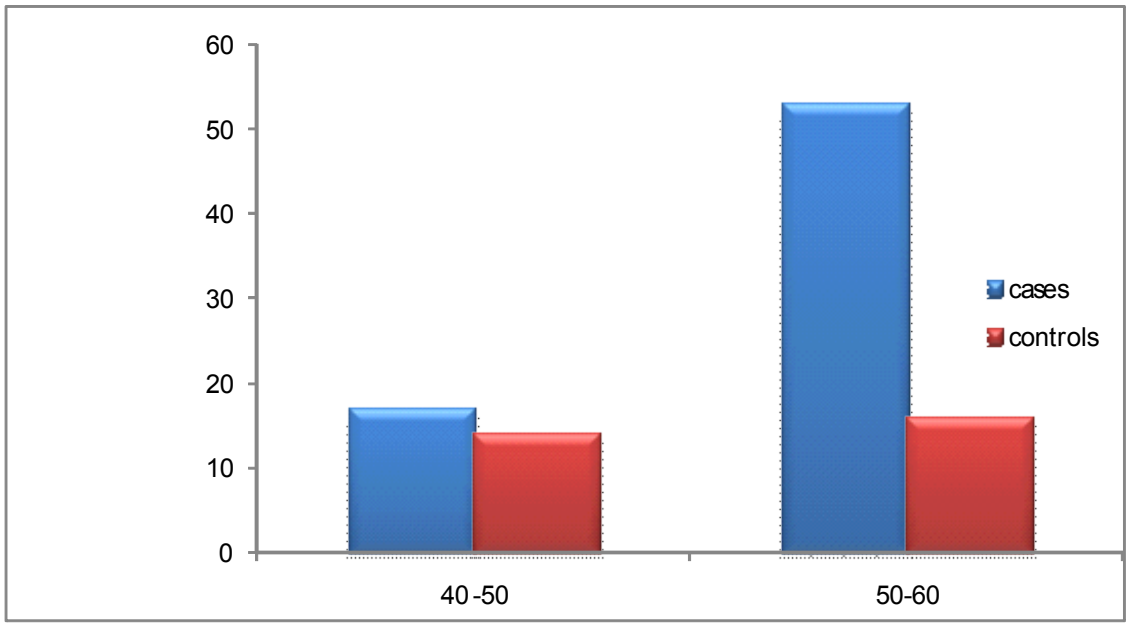
Table IV

Distribution of cases and controls in relation to age

Age group	Cases		Control	
	No	%	No	%
41-50	17	24	14	46
51-60	53	76	16	54
Mean	53.7		51.9	
S.D	4.88		4.89	

FIG 1

Distribution of cases and controls according to age



Among the cases the the mean and standard deviation of age for stage I and stage II hypertensives were 52.9± 5.12 and 54.2± 4.72 respectively .so there is no difference statistically between two groups statistically with reference to age.

TABLE - V

	STAGE I	STAGE II
MEAN	52.9	54.2
STANDARD DEVIATION	5.12	4.72

Distribution of cases and controls in relation to gender

Among cases there were 38 males and 32 females. Among controls there were 20 males and 10 females. This is shown in TABLE-VI given below

TABLE VI

Sex	Cases		Control	
	No	%	No	%
Males	38	54	20	67
Females	32	46	10	33
Total	70	100	30	100

The group of cases comprises of both stage I and stage II hypertensives. Among stage I hypertensive cases there were 14 males and 12 females. Among stage II hypertensives there were 24 males and 20 females .This is shown in TABLE-VII.

Distribution of stage I and stage II hypertensives in relation to gender

TABLE VII

Sex	STAGE I		STAGE II	
	No	%	No	%
Males	14	54	24	55
Females	12	46	20	45
Total	26	100	44	100

Analysis of cases and controls with respect to Body mass index

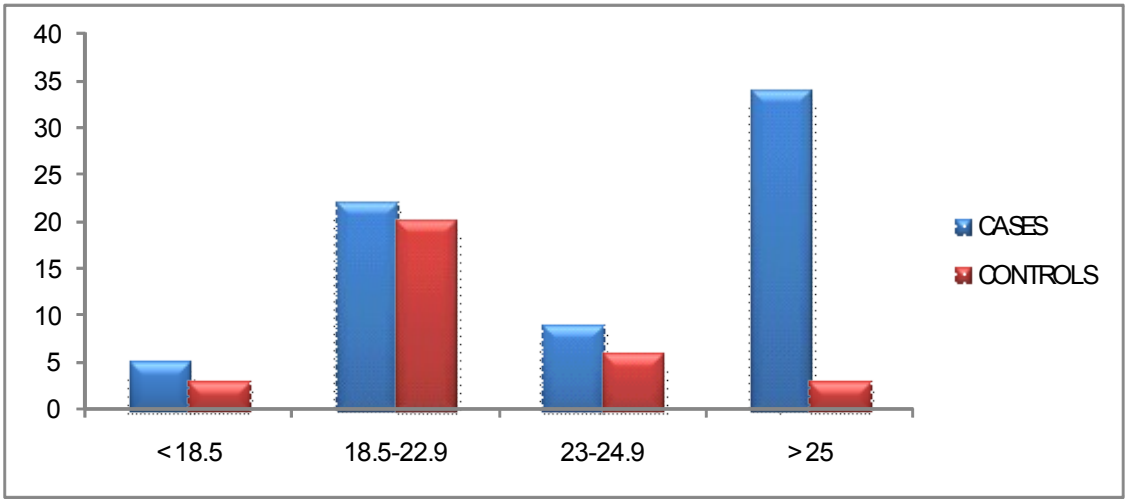
49 % of cases were obese whereas among control group only 3% were obese. The details are given in TABLE VIII (FIG-2) shown below

TABLE VIII

B.M.I	Cases		Control	
	No	%	No	%
Underweight < 18.5	5	7	3	10
Normal 18.5-22.9	22	31	20	67
Overweight 23-24.9	9	13	6	20
Obese > 25	34	49	3	3
Total	70	100	30	100

FIG -2

Distribution of cases and controls with respect to their Body mass



Distribution of stage I and stage II hypertensives with respect to Body mass index [B.M.I]. (Kg/m^2)

TABLE IX

B.M.I	stage I		stage II	
	No	%	No	%
Underweight < 18.5	1	4	4	9
Normal 18.5-22.9	11	42	11	25
Overweight 23-24.9	3	12	6	14
Obese > 25	11	42	23	52
Total	26	100	44	100

Body Mass Index [B.M.I] among stage I and stage II hypertensives does not differ significantly (TABLE-IX).

Body Mass Index [B.M.I] among cases and control

The mean and standard deviation of B.M.I among cases and control were shown below TABLE X shown below (FIG-3)

TABLE X

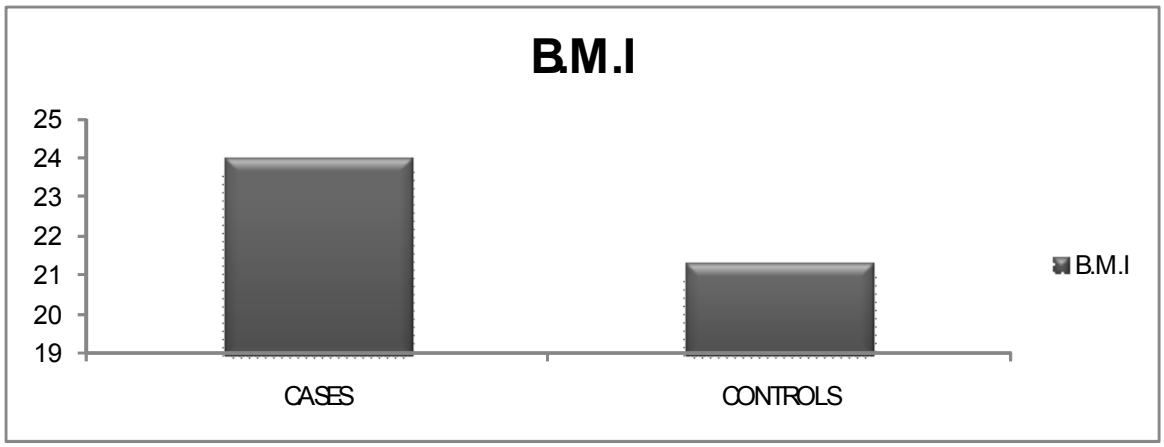
B.M.I	CASES	CONTROL
MEAN	24	21.3
S.D	3.15	2.14

P value = 0.00004

This table shows that there is a statistically significant difference in B.M.I among cases and control.

FIG-3

Body Mass Index [B.M.I] among cases and control



Body Mass Index [BMI] among stage I and stage II hypertensives

TABLE XI

B.M.I	STAGE I	STAGE II
MEAN	23.91	24.15
S.D	3.41	3.61

P VALUE = 0.78

Though there appears to be a difference in Body Mass Index [BMI] between stage I and stage II hypertensives it was not statistically significant. (TABLE – XI).

Body Mass Index [B.M.I] among cases controls with respect to gender

The table XII below shows the Body Mass Index [B.M.I] among the cases and controls with respect to gender.

TABLE XII

B.M.I	Case	Control	P VALUE
Male	24.19 ± 3.62	20.90 ± 2.07	0.0004
Female	23.92 ± 3.44	22.2 ± 2.1	0.14

The mean of Body Mass Index among male cases are 24.19 which is significantly higher statistically compared male controls. There is no statistically significant difference in Body Mass Index [B.M.I] among female cases and controls.

Analysis of presenting symptoms

The cases both stage I and stage II hypertensives were analysed with respect to their presenting symptom. This is shown in TABLE XIII given below

TABLE XIII

Symptoms	stage I		stage II	
	No	%	No	%
Giddiness	12	46	12	27
Headache	7	27	16	36
Chest pain	3	12	6	14
Palpitation	4	5	8	18

Dyspnoea	0	0	2	5
Total	26	100	44	100

Headache and giddiness were the more common symptoms of the cases in our study both stage I and stage II hypertensives.

Distribution of systolic and diastolic blood pressure

As the raised blood pressure was the nature of the disease taken in to study it was not statistically analysed. The distribution of systolic and diastolic blood pressure among cases and controls is shown in TABLE XIV given below

TABLE XIV

BLOOD PRESSURE	CASES MEAN ± S.D	CONTROL MEAN ± S.D
Systolic	167.82±18.50	106.9±6.46
Diastolic	104.51±8.51	71.6±4.32

Distribution of systolic and diastolic blood pressure among stage I and stage II hypertensives

The distribution of systolic and diastolic blood pressure among stage I and stage II hypertensives is shown in TABLE XV below

TABLE XV

B.M.I	SYSTOLIC B.P MEAN ± S.D	DIASTOLIC B.P MEAN ± S.D
STAGE I	178.5 ±14.43	109.81 ±6.03
STAGE II	149.77 ±6.92	95.54 ±1.985

Risk factors among cases and controls

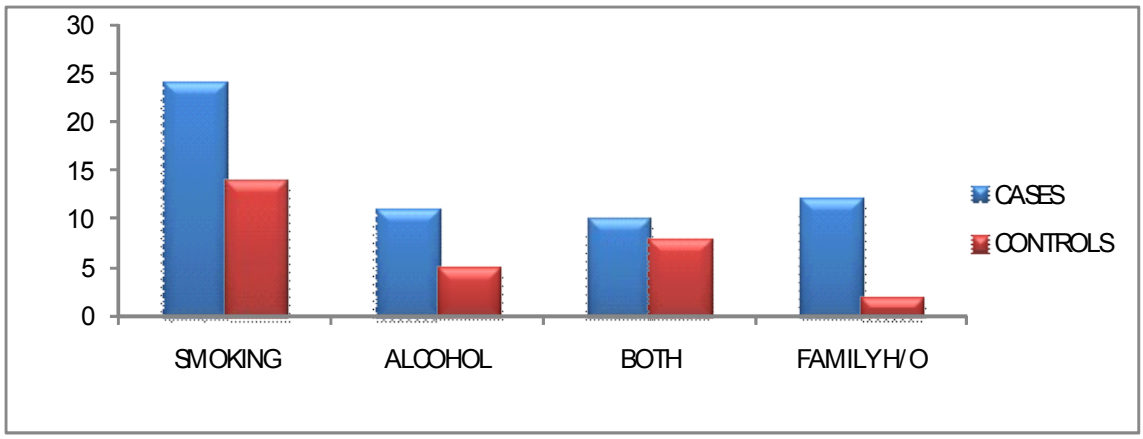
TABLE XVI

	smoking		Alcohol		both	Family history	
	yes	no	yes	No		Yes	No
Cases	24	46	11	59	10	12	58
Controls	14	16	5	24	5	2	28

The risk factors like smoking alcoholism and family history of any cardiovascular disease were equally seen among cases and controls this is shown in TABLE XVI above (FIG – 4)

FIG – 4

Risk factors among cases and controls



Risk factors among stage I and stage II hypertensives

TABLE XVII

	smoking		Alcohol		Both	Family history	
	yes	no	yes	No		Yes	No
stage I	10	16	4	21	4	3	23
stage II	14	30	7	38	5	9	35

The above TABLE XVII shows the prevalence of risk factors among stage I and stage II hypertensives .This shows no significant difference.

Serum sodium among cases and controls

The Table XVIII below shows the mean and standard deviation of serum sodium among cases and control. (FIG – 5)

TABLE XVIII

Serum sodium	Cases	Controls	
Mean	141.80	138.96	P = 0.00001
S.D	2.91	2.59	

The serum sodium level was significantly more among hypertensives than controls. (P value = 0.00001)

Serum sodium among stage I and stage II hypertensives

The Table XIX shows the mean and standard deviation of serum sodium among stage I and stage II hypertensives (FIG – 6)

TABLE XIX

Serum sodium	stage I	stage II	
Mean	140.73	142.43	P = 0.017
s.d	2.539	2.96	

FIG – 5

Serum sodium among cases and controls

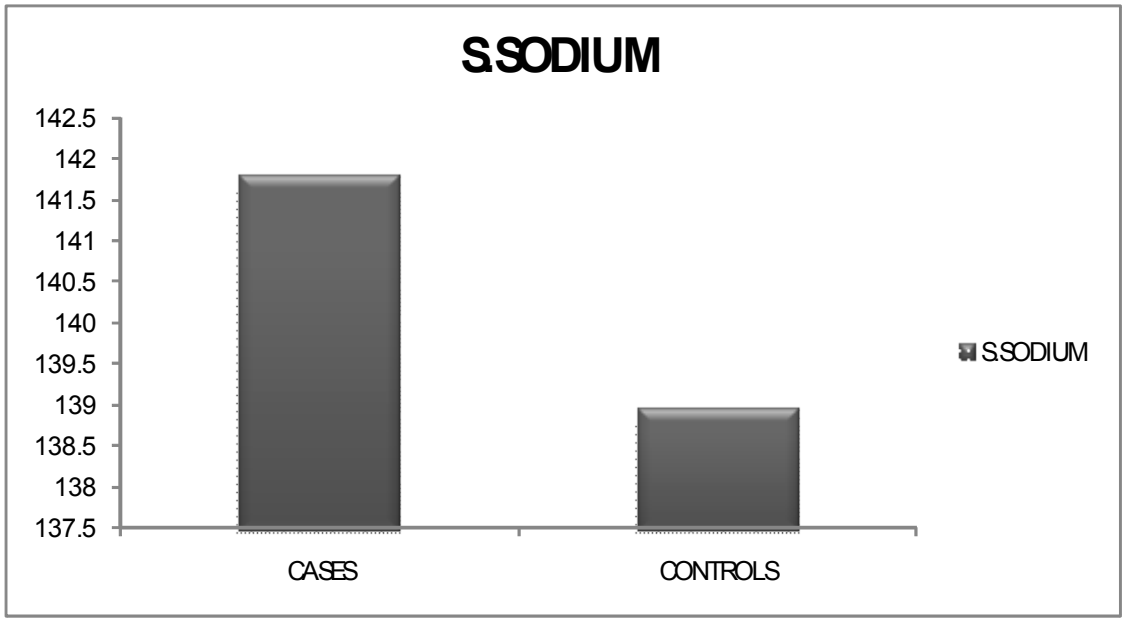
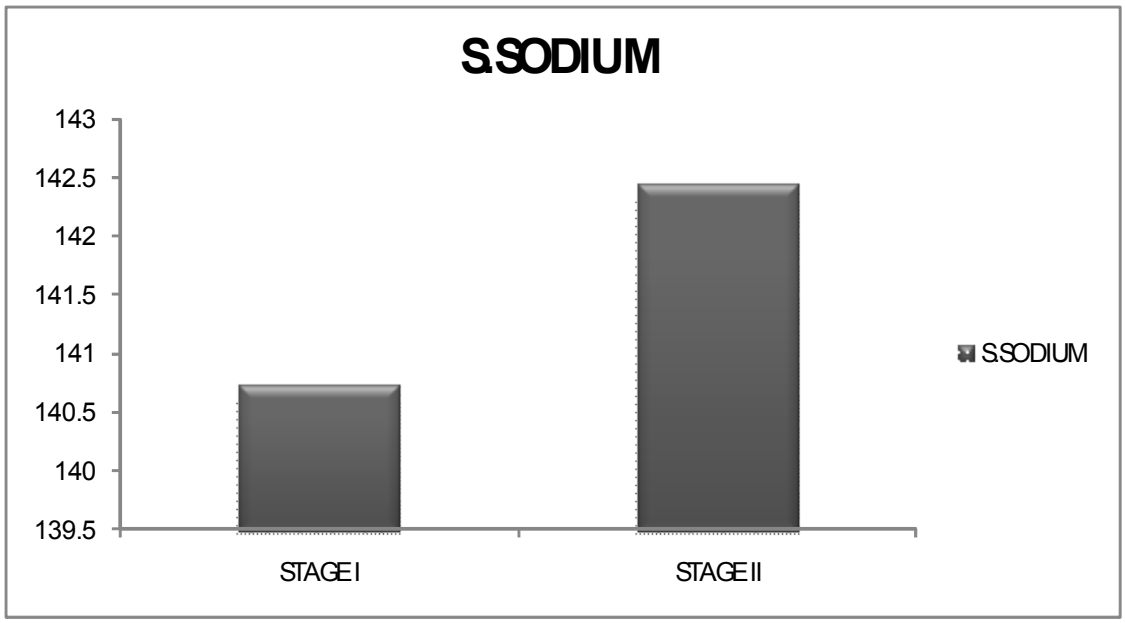


FIG – 6

Sodium among stage I and stage II hypertensives



The serum sodium level was significantly higher among stage I hypertensives than stage II hypertensives. (P value = 0.017).

Serum sodium among males and females

TABLE XX

Serum sodium	Cases	Controls
Males	141.84±3.063	138.95±2.982
Females	141.75±2.782	139±1.7
P value	0.89	0.96

The table above shows the serum sodium values among males and females in both cases and control group. The statistical analysis

showed no significant difference in serum sodium levels among cases and controls with respect to their gender.

Serum potassium among cases and controls

The table below shows the mean and standard deviation of serum potassium among cases and control (FIG -7).

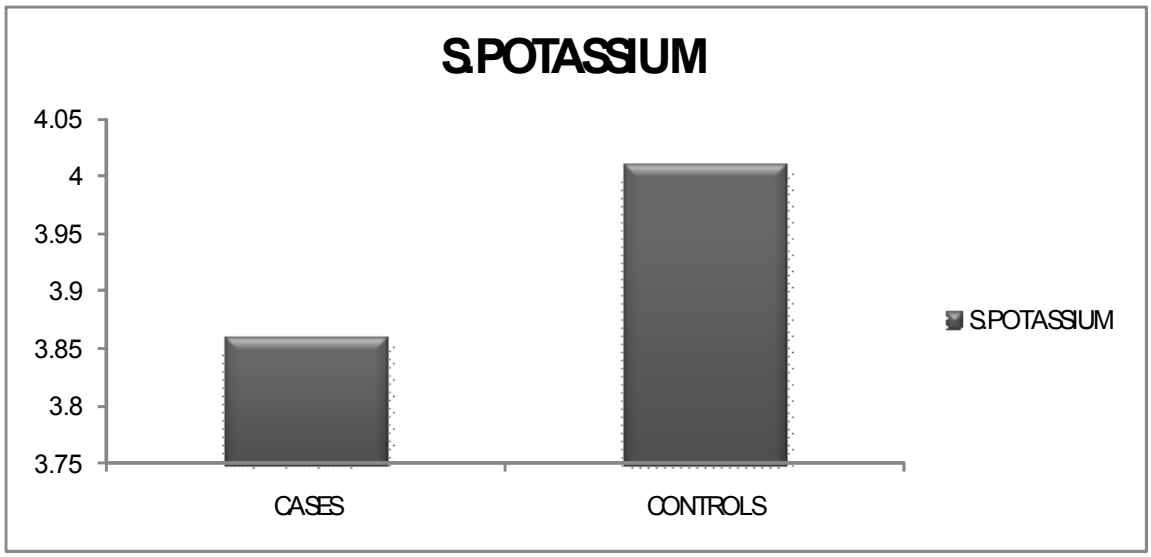
TABLE XXI

Serum potassium	Cases	Controls	
Mean	3.86	4.013	P= 0.00004
S.D	0.197	0.196	

The serum potassium level was significantly more among controls than hypertensives.(P VALUE = 0.00004)

FIG – 7

Serum potassium among cases and controls



S. potassium among stage I and stage II hypertensives

The table shows the mean and standard deviation of serum potassium among stage I and stage II hypertensives.

TABLE XXII

Serum Potassium	Stage I	Stage II	
Mean	3.86	3.85	P = 0.83
S.D	0.22	0.18	

S. Potassium among males and females

This Table XXIII shows the serum sodium values among males and females in both cases and control group. The statistical

analysis showed no significant difference in serum potassium levels among cases and controls with respect to their gender.

TABLE XXIII

Serum potassium	Case	Control
Male	3.86 ± 0.19	4.05±0.20
Female	3.86±0.20	3.95±0.17
P value	0.98	0.21

Twenty four hour Urine sodium excretion among cases and controls

The Table XXIV below shows the mean and standard deviation of urine sodium among cases and control. (FIG - 8)

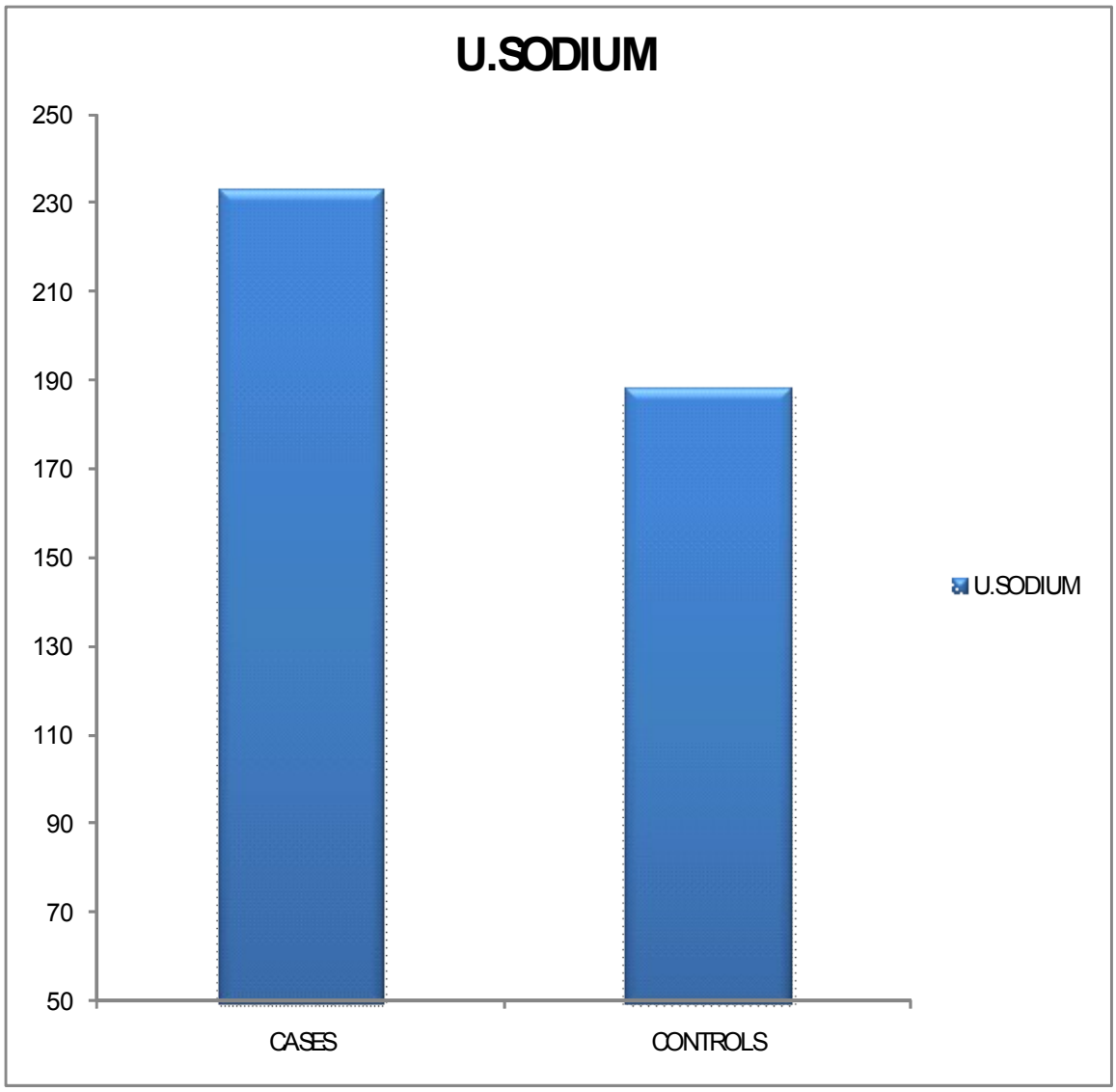
TABLE XXIV

Urine sodium	Cases	Controls	
Mean	232.89	188.10	P=0.001
S.D	70.15	47.04	

The main aim of our study is to study twenty four hour Urine sodium among cases and controls. The urine sodium level was significantly more among hypertensives than controls. (P = 0.001)

FIG -8

Twenty four hour Urine sodium excretion among cases and controls



Twenty four hour Urine sodium excretion among Stage I and Stage II hypertensives.

The Table XXV below shows the mean and standard deviation of urine sodium among Stage I and Stage II hypertensives (FIG – 9)

FIG – 9

Twenty four hour Urine sodium excretion among Stage I and Stage II hypertensives.

TABLE XXV

Urine sodium	Stage I	Stage II	
Mean	208.92	247.04	P value =0.027
S.D	59.64	72.64	

The urine sodium level was significantly higher among stage I hypertensives than stage II hypertensives.

Twenty four hour Urine sodium excretion with respect to gender

This Table XXVI shows the serum sodium values among males and females in both cases and control group .The statistical analysis showed no significant difference in serum sodium levels among cases and controls with respect to their gender.

TABLE XXVI

Urine sodium	Case	Control
Male	231.42 ± 70.67	178.95 ± 48.36
Female	234.72 ± 70.62	206.40 ± 40.44
P value	0.84	0.33

Twenty four hour urine potassium excretion among cases and controls

The Table XXVII below shows the mean and standard deviation of urine potassium among cases and control.

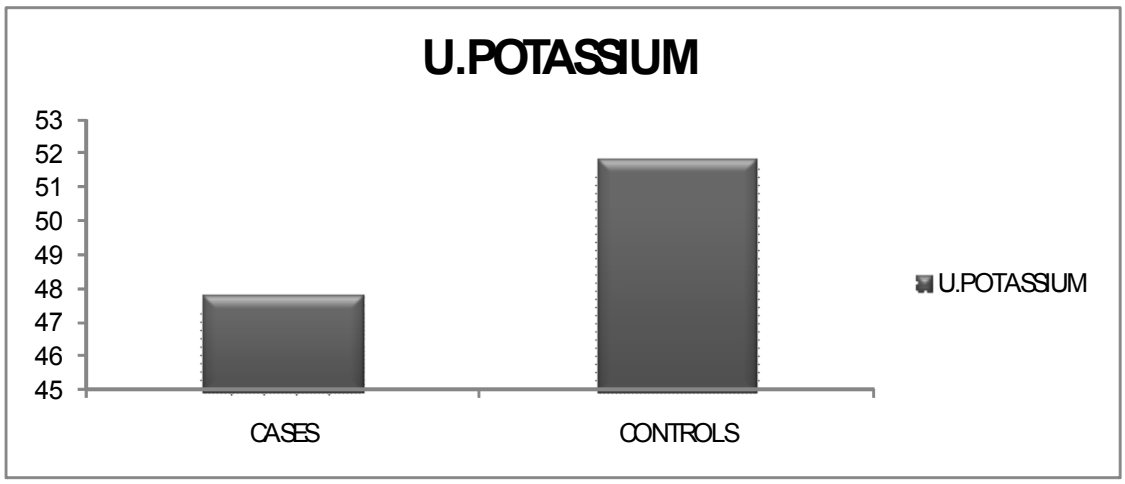
TABLE XXVII

Urine potassium	Cases	Control	
Mean	45.76	51.83	P Value = 0.13
s.d	18.91	17.39	

The urine potassium level was not significantly lower among hypertensives than controls.

FIG -10

Twenty four hour urine potassium excretion among cases and controls



Twenty four hour urine potassium excretion among stage I and stage II hypertensives

The urine potassium level was not significantly higher among stage I hypertensives than stage II hypertensives as shown in table below

TABLE XXVIII

Urine potassium	Stage I	Stage II	
Mean	46.73	45.18	
s.d	20.74	17.96	P value = 0.74

Twenty four hour urine potassium excretion with respect to gender

This Table XXIX shows the urine potassium values among males and females in both cases and control group. The statistical analysis showed no significant difference in urine potassium levels among cases and controls with respect to their gender.

TABLE XXIX

Urine potassium	Case	Control
Males	46.10 ± 19.24	50 ± 18.63
Females	45.34 ± 18.81	45.16 ± 18.41
P value	0.86	0.50

Analysis of sodium and potassium in serum and urine with respect to their Body Mass Index [B.M.I]

The Table below shows the mean and standard deviation of sodium and potassium in serum and urine with respect to their Body Mass Index [B.M.I] (FIG 19 AND FIG 20).

TABLE XXX

PARAMETER	Low B.M.I	High B.M.I	P value
Serum sodium	140.74 ± 2.93	142.46 ± 2.74	0.01
Serum potassium	3.88 ± 0.22	3.84 ± 0.18	0.53
Urine sodium	227.03 ± 71.70	236.56 ± 69.75	0.58
Urine potassium	46.48 ± 19.83	45.30 ± 18.52	0.80

For this analysis we compare cases with body mass index < 23(low B.M.I) with cases having body mass index ≥ 23(high B.M.I).

The statistical analysis of sodium and potassium levels in serum and urine with respect to their body mass index [B.M.I] showed that there is significantly high serum sodium in cases with high body mass index than cases with low body mass index. The levels of serum potassium, urine sodium and urine potassium does not differ significantly among cases with higher and lower body mass index.

Analysis of sodium and potassium in serum and urine with respect to their body mass index among controls

The table below shows the mean and standard deviation of sodium and potassium levels in serum and urine with respect to their B.M.I among controls.

TABLE XXXI

PARAMETER	Low B.M.I	High B.M.I	P value
Serum sodium	138.60=-2.55	140.14=-0.20	0.17
Serum potassium	4.04=-0.20	3.93=-0.16	0.16
Urine sodium	180.13=-42.49	200.28=-60.44	0.32
Urine potassium	52.39=-15.95	50=-22.86	0.75

There was no statistically significant difference in sodium and potassium levels in serum and urine among controls.

DISCUSSION

Essential hypertension comprises 90% of all hypertensive patients.[1] various factors have been responsible for perpetuation or causation of hypertension. In addition to hereditary predisposition, high sodium intake and

lower potassium intake and excretion may also contribute to the development of hypertension which has been proved.[3] Excretion of sodium and potassium depend on their intake. Positive correlation between salt excretion and blood pressure has been proved by some and disproved by other recent studies.[66, 67]

Present study was conducted to clear this controversy in a population who have habit of excessive salt intake in the form of sea foods like dry fish, pickles and even the water they use have high sodium due to nearness to sea.

In the Chennai Urban Rural Epidemiology Study (CURES) [68] an ongoing population based study on a representative population of Chennai city in southern India, mean dietary salt intake (8.5 g/d) in the population was higher than the recommended by the World Health Organization (<5g/d).

Our study population is also from Chennai city. In our study we analysed serum sodium, serum potassium, urine sodium and urine potassium (twenty four hour collection) among newly diagnosed hypertensives. Primary aim of our study is to compare 24 hour urine sodium excretion in hypertensives and normotensives and also to study with respect to their body mass index and stages of hypertension.

The varsity of hypertension among those consuming large amount those consuming large amount of salt may probably be related to chronic adaptation of body system towards renal clearance of sodium.

SERUM SODIUM AMONG HYPERTENSIVES

In our study serum sodium was estimated in control and study groups and compared between them. Results were compared with other studies. Serum sodium was higher in the hypertensive group than the control group even through both were within the normal range.

The mean and standard deviation of serum sodium among cases was 141.80 ± 2.91 while in the control group was 138.96 ± 2.59 respectively [p=0.00001]. Our study was supported by RA Jan, et al (2006)[69], Srinagar, Kashmir. In this study 135 hypertension patients and equal number of age and sex matched healthy controls were taken for the study. Serum sodium in the hypertension group was 140 ±2.90 while in the control group it was found to be 138.5±1.12. Serum sodium was higher in the hypertension group than the control group and considered to be a factor responsible for causation of hypertension.

In our study the mean and standard deviation of serum sodium among stage 1 and stage 2 hypertensives were 140.73 ± 2.54 and 142.43 ± 2.96 respectively[p=0.017].The serum sodium level was significantly higher among stage II hypertensives than stage I hypertensives. This was also supported by RA Jan, et al (2006), Srinagar, Kashmir.

A study was carried out by Lever et al of arterial pressure and body content of electrolytes in 91 patients with essential hypertension and 121 normal control [70].Plasma and exchangeable sodium was found to be positively correlated with arterial pressure in the patients.

In another study conducted by Williams et al, they studied the relationship of body sodium, chlorine and potassium in 30 patients with essential hypertension. They found that a positive correlation exists between serum sodium and blood pressure in this study group[71].

In another study conducted by Bulpitt, two thousand three hundred and 28 men and 1496 women between the ages of 35 and 64 years were screened for hypertensive and other plasma sodium and its concentration measured. It was found that plasma sodium was positively related to that of blood pressure and an increased in serum sodium of 1mmol/l was associated with an increase of 1mmol hg in both men and women[72].

SERUM POTASSIUM AMONG HYPERTENSIVES

In our study serum potassium was estimated in control and study groups and compared between them. Serum potassium was found to be lower in the hypertensive group when compared with control group even though both were within normal range.

The mean serum potassium in study group was 3.86±0.197.The mean potassium in the control group was 4.013±0.196. [p=0.00004]. This is statistically significant. There was no significant difference among the 2 stages of hypertension.

A study was carried out by Bulpitt et al among two thousand three hundred and twenty eight men and 1496 women between the ages of 35 and 64 years, a decrease in plasma potassium of 1mmol was associated with an increase in systolic pressure in women of 7mmHg (p less than 0.001) and diastolic pressure of 4mmHg (p less than 0.001). In men corresponding increases were 4mmHg ($p<0.01$) and 2mmHg ($p<0.05$)[72]

Similarly in a study conducted by Lever et al [70] in 121 normal subjects and 91 hypertensive patients it was shown that plasma exchangeable on a total body potassium correlated inversely with arterial pressure in the patients. They suggested the following theory as the cause of essential hypertension.

In the early stages of the disease blood pressure is raised by an abnormal process related more closely to potassium than to sodium. A renal lesion develops later possibly as a consequence of the hypertension. This lesion is characterised by resetting of pressure natriuresis and is manifest by an abnormal relation between body sodium and arterial pressure and by susceptibility to increased dietary sodium intake.

Similarly a study was conducted at national institute of public health and environmental protection, Bilthoven, the Netherlands, the relationship between the serum cations sodium, potassium, calcium, magnesium and blood pressure were investigated in a population based sample of 182 Dutch persons

aged 20-59 years. In the combined analysis, a weak inverse relationship was found between serum potassium and diastolic blood pressure. [73]

In an another study carried out at Karachi, Pakistan 30 hypertension diabetic patients and equal number of age and sex matched controls were taken for the study.he mean serum potassium was 4.59 mmol/l among the control group[74].

To investigate the role of potassium on blood pressure luft et al conducted a study among 431 normotensive and 478 hypertensive subjects. They observed an inverse relationship between serum potassium and blood pressure supporting our study.[75]

24 HOUR URINE SODIUM AND HYPERTENSION

In our study the mean and standard deviation for 24 hour urine sodium in cases and control were232.89±70.15 and188.10 ± 47.04 [p= 0.001]. The mean and standard deviation for 24 hour urine sodium in stage 1 and stage 2 hypertensives were 208.92± 59.64 and 247.04 ±72.64[p=0.027].

So there was a statistically significant increase in sodium excretion among hypertensives which was more with increased blood pressure [more in stage II than stage I hypertensives]

This observation was supported by RA Jan, et al[69] (2006), Srinagar, Kashmir. In that study the mean and standard deviation for 24 hour urine sodium excretion was very high among both hypertensives and normal

subjects 424 ± 150.50 and 337 ± 121.50 respectively ($p < .001$).This was due to high intake of salt tea in that population (average 27 g/day) consistent with a studies done in Japan (average 11-27 g/day).[76,77]

Excessive 24 hr urinary sodium excretion has been seen in other studies as well.[7879]

In a study conducted by Tuomilehto et al[80], a 24-hour urine collection was carried out during a cardiovascular survey in eastern Finland. The study population comprised 148 hypertensive subjects in a random sample of the middle-aged population and 86 normotensive controls. The mean sodium excretion was 197 mmol /24h in normotensive and hypertensive men, 179 mmol/24h in normotensive women, and 174 mmol /24h in hypertensive women.The results of this study show that the average level of salt intake in a Finnish population is high, and so is the sodium: potassium molar ratio. Although there was no correlation between sodium excretion and blood pressure levels, it is known that in this population the average blood pressure level is high and cardiovascular disease extremely frequent.

Intersalt[3] found significant positive relations between 24 hour urinary sodium excretion and systolic and diastolic blood pressure in individual participants and between individual urinary sodium to potassium ratios and blood pressure.

Statistically significant increased 24 hr excretion of sodium in hypertensives in our study was possibly responsible in causation of hypertension which was in agreement to various other studies.[81-83]

Sensitivity to hypertensive effect of sodium may be genetically determined which may be also operative in our cases as well.[84] It is also supported by fact that hypertension decreased by low salt intake and hypertension is absent or rare in population with low salt intake as is the antihypertensive effect of thiazides due to loss of sodium and extra cellular fluid.[83, 85, 86]

24 HOUR URINE POTASSIUM AND HYPERTENSION

Though the main aim of our study is to compare 24 hour urine sodium excretion in hypertensives and controls we also analysed 24 hour urine potassium levels in hypertensives and normal.

In our study the mean and standard deviation for 24 hour urine potassium in cases and control were 46.75±18.91 and 51.83±17.39 [p= 0.13]. the mean and standard deviation for 24 hour urine potassium in stage 1 and stage 2 hypertensives were 46.73±20.74 and 45.18±17.96 [p=0.74].

In both hypertensives and normotensives, average 24 hour urinary excretion of potassium was lower because of low intake of potassium in this part of world which might also contribute to hypertension as has also been seen by Menealy and Belarbee in 1976.[81]

Low potassium intake leading to increase blood pressure and vice versa is confirmed by various other studies independent of sodium excretion.[81],[87] Recently there was evidence of additive effect of high sodium intake and low potassium intake in causation of hypertension which may be operative in our hypertensive group as well.

BODY MASS INDEX AND HYPERTENSION.

In our study the mean body mass index among the study group was 24 ± 3.15 and among the control group was 21.3±2.14.the "p" value was 0.00004.This shows that overweight and obesity also plays a role in the development of essential hypertension.

This was supported by a study conducted by Stamler[51]. They showed that the hypertension is about six times more common in obese than it is in lean subjects. The present study concurs with above observation.

Similarly a study conducted by Huang stated that even a small amount of weight gain is associated with a marked increase in the incidence of hypertension[88]. This study showed a positive correlation between body mass index and blood pressure which supported our study.

In INTERSALT[89], the relationship between body masss index(kg/m^2) and blood pressure was studied in 10,079 men and women aged 20-59, sampled from 52 centres around the world, based on a standardized

protocol with central training of observers, a central laboratory and extensive quality control. Body mass index and blood pressure relationships were first studied in men and women within each centre and results of these regression analyses were then pooled for all 52 centres, with adjustment for age, alcohol intake, smoking, sodium and potassium excretion, body mass index was positively associated with systolic blood pressure in some centres and diastolic blood pressure in some centres. In further analyses across centres median body mass index related significantly to median systolic blood pressure, median diastolic pressure and prevalence of hypertension in both men and women. Body mass index was related to the slopes of systolic and diastolic blood pressure with age in women but not in men.

In our study the mean and standard deviation for serum sodium levels among cases with low and high body mass index were 140.74±2.93 and142.46 ±2.74 respectively which was statistically significant[p=0.01]. The mean and standard deviation of urine sodium among cases with low and high body mass index were 227.03±71.70 and 236.56 ± 69.75 respectively. Although sodium excretion seems to be high in cases with higher body mass index this was not statistically significant [p=0.58].

So raised body mass index have played a significant role in increased serum sodium seen in hypertensives, but the role played by body mass index in increased sodium excretion seen among hypertensives were insignificant.

A study conducted by Jan et al [69]observed significant impact of BMI on 24 hr Na+ excretion in both groups with increased sodium excretion with high BMI while there was no association of BMI with K+ excretion in either group.

Relationship of BMI with Na+ output and hypertension has been proved by Sampson *et al* in 1978[90]. BMI showed no significant influence on Na+ excretion in hypertension patients in our study but while estimation BMI should always be considered.

Age modifies a number of factors i.e., GFR, renal hemodynamics and responsiveness of renin angiotensin aldosterone system confirmed by Norma K Hollenberg as well. Age influences the capacity of kidneys to conserve sodium, so age related changes must be considered while estimations.[91]

In our study we did not attempt to study the age related differences in sodium excretion because our subjects fall within a smaller group 40 to 60 years.

Also in our study there was no statistically significant difference in sodium excretion among males and females.

CONCLUSION

The following conclusions were derived from our study.

1) Serum sodium was significantly more among hypertensives and was also correlated positively with the level of blood pressure. It was independent of associated risk factors and gender.

2) Serum potassium was significantly less among hypertensives and it correlated negatively with blood pressure.

3) Markedly increased 24 hour sodium excretion and low potassium excretion in hypertensive group indicates a very high intake of sodium and low intake of dietary potassium both playing significant individual as well as additive role in causation or perpetuation of hypertension.

4) Body mass index has direct bearing on blood pressure but not on sodium excretion.

5) In view of significant changes in simple electrolyte levels (sodium & potassium) among hypertensive population, community must be motivated to reduce their intake of common salt and encouraged to consume potassium rich nutrients-diets as a form of prevention for essential hypertension.

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PROFORMA

NAME : AGE: SEX: F/M

DIET : V/NV

ADDRESS: OCCUPATION:

SYMPTOMS:

Headache	Oliguria	Giddiness
Puffiness of face	Blurring of vision	Epistaxis
Swelling of legs	Anorexia	Chest pain
Vomiting/hiccups	Palpitation	Easy fatiguability
Dyspnoea	Polydipsia	Polyuria

PAST HISTORY

Diabetes mellitus	Angina/MI	Heart failure
Renal disorders	PVD	Stroke

PERSONAL HISTORY

Smoking	Alcohol	Drug abuse

FAMILY HISTORY

Hypertension	Diabetes mellitus

ANTHROPOMETERY

Wt (kg)	Ht (cm)	B.M.I (Kg/m^2)

Hip (cm) waist (cm) WHR

GENERAL EXAMINATION

Fundus

Pulse rate

Blood pressure (mm/hg) = 1) 2) 3)

Mean of 3 readings =

Supine Sitting

Staging of hypertension

Pedal edema

SYSTEMIC EXAMINATION

C.V.S =

R.S =

P/A =

INVESTIGATION

URINE

Albumin

Sugar

Deposits

Blood glucose

Serum urea

Serum Creatinine

Serum sodium =

Serum potassium =

TWENTY FOUR HOUR URINE

Volume =

Creatinine=

Sodium =

Potassium =

Protein =

ECG

ULTRASOUND ABDOMEN

CHEST X-RAY PA VIEW

MASTER CHART

S.NO	NAME	AGE	SEX	SYMPTOM	P/H	SMOKING	ALCOHOL	F/H	B.M.I Kg/m²	S.B.P mm/Hg	D.B.P mm/Hg	FUNDUS	S.ε	B.GLUCOSE	B.UREA	S.CREATINE	S.SODIUM	S.POTASSIUM	E.C.G	C.X.R	USG.ABD
1	PANDURANGAN	58	M	1	N	Y	Y	N	26.7	154	94	N	N	85	38	1.1	138	3.8	H	N	N
2	ARPUTHAM	54	F	2	N	N	N	N	25.8	156	110	N	N	78	26	1	145	3.8	N	N	N
3	MALLIKA	55	F	2	N	N	N	N	25.9	148	94	N	N	80	26	0.9	140	4.3	N	N	N
4	SAROJA	58	F	1	N	N	N	N	22.4	154	98	N	N	82	36	0.8	142	3.9	N	N	N
5	BALARAMAN	52	M	2	N	Y	N	B	22.1	156	94	N	N	132	54	1	143	4.3	N	N	N
6	VIJAYA	60	F	3	D	N	N	D	28.7	158	98	N	N	138	52	1.1	144	3.6	N	N	N
7	DAMODARAN	52	M	4	N	Y	N	N	23.8	156	98	N	N	86	38	0.9	142	4.2	L	N	N
8	RAJENDRAN	49	M	1	N	Y	Y	N	29.5	158	96	N	N	70	24	0.8	143	3.8	N	N	N
9	IQBAL	59	M	2	N	N	N	N	26.9	142	98	I	N	72	32	0.8	142	3.6	N	N	N
10	ELUMALAI	46	M	4	N	Y	N	Y	17.3	164	100	N	N	76	24	0.9	137	3.7	N	N	N
11	JAYARAMAN	57	M	2	D	Y	Y	N	21.1	182	120	N	N	134	48	0.9	145	3.9	N	N	N
12	SELVAKUMAR	59	M	2	N	N	N	N	25.4	180	110	N	N	78	24	0.9	143	3.6	N	N	N
13	NAGOORKANI	53	F	4	N	N	N	N	25.8	186	110	N	N	68	44	0.8	144	3.8	L	N	N
14	AMARAN	56	M	3	N	Y	N	Y	23.8	186	104	N	N	76	24	0.6	136	3.7	L	C	N

15	MUNIYAMMAL	55	F	1	N	N	N	N	24.2	130	96	N	N	90	35	0.9	142	4.2	N	N	N
16	JAYALAKSHMI	46	F	1	N	N	N	N	25.6	154	96	N	N	98	32	1	143	4	N	N	N
17	MUTHU	48	M	1	N	N	N	N	20.9	154	96	N	N	88	34	0.9	139	3.7	N	N	N
18	MARIYAMMAL	46	F	4	N	N	N	N	18.1	176	104	N	N	65	29	1	144	3.7	N	N	N

S.NO	NAME	U.VOLUME(ml)	U.PROTEIN (mg/24 hrs)	U.CREATININE (mg/24hrs)	U.SODIUM (mg/24hrs)	U.POTASSIUM (mg/24 hrs)
1	PANDURANGAN	1650	67	1291	240	76
2	ARPUTHAM	1600	67	1454	325	75
3	MALLIKA	1500	77	1133	130	41
4	SAROJA	1450	54	1064	252	67
5	BALARAMAN	1450	48	1527	254	21
6	VIJAYA	1750	50	929	240	74
7	DAMODARAN	1500	64	1544	138	20
8	RAJENDRAN	1250	64	1705	267	56
9	IQBAL	1150	75	1395	232	68
10	ELUMALAI	1200	56	1878	172	59
11	JAYARAMAN	1650	71	1429	224	56
12	SELVAKUMAR	1350	49	1701	173	26
13	NAGOORKANI	1300	37	820	166	39

14	AMARAN	1350		52		1310		284		41										
15	MUNIYAMMAL	1200		31		900		186		61										
16	JAYALAKSHMI	1300		61		937		156		26										
17	MUTHU	1650		60		1757		264		43										
18	MARIYAMMAL	1250		68		1171		125		59										

S.NO	NAME	AGE	SEX	SYMPTOM	P/H	SMOKING	ALCOHOL	F/H	B.M.I Kg/m²	S.B.P mm/Hg	D.B.P mm/Hg	FUNDUS	S. ш	B.GLUCOS ı	B.UREA	S.CREATININ ı	S.SODIUM	S.POTASSIU ..	E.C.G	C.X.R	USG.ABD
19	SAROJA	45	F	5	N	N	N	N	23.8	176	109	N	N	83	22	0.7	144	4	N	N	N
20	SARIKA	44	F	2	N	N	N	N	23.1	174	106	N	N	68	28	0.8	141	3.9	N	N	N
21	SARASA	48	F	4	N	N	N	N	25.2	156	94	N	N	90	34	0.9	142	3.6	N	N	N
22	AMBIKA	56	F	1	N	N	N	N	26.6	178	106	N	N	77	22	0.8	136	4.2	N	N	N
23	ALAGAMMA	59	F	4	N	N	N	D	26.6	188	100	N	N	76	22	0.9	141	3.6	N	N	N
24	MUTHU	55	M	1	N	Y	N	N	17.4	140	92	N	N	80	36	0.8	140	3.6	N	N	N
25	SELVAM	51	M	1	N	N	N	N	18.1	158	114	N	N	81	25	0.9	143	3.8	N	N	N
26	MOORTHY	57	M	1	N	Y	Y	Y	20.2	158	110	N	N	72	34	08	140	4.2	H	N	N
27	AYISHA	57	F	1	N	N	N	N	17.3	162	102	N	N	88	36	0.7	143	3.7	N	N	N
28	BASHEERA	57	F	1	N	N	N	N	25.4	178	100	N	N	86	26	0.8	143	3.6	N	N	N

29	KAMATCHI	60	F	2	N	N	N	Y	23.8	180	108	N	N	88	28	1.2	143	3.7	N	N	N
30	RAJA	60	M	4	N	Y	N	N	23.1	188	106	N	N	82	34	1.1	141	3.9	N	N	N
31	GOVINDASAMY	56	M	1	N	Y	Y	N	26.6	168	109	N	N	83	20	0.9	144	3.6	N	N	N
32	KANNAN	55	M	1	N	N	N	N	20.4	168	120	N	N	69	28	0.9	139	3.9	N	N	N
33	KAMALA	52	F	3	N	N	N	Y	26.8	200	120	N	N	96	22	0.8	143	3.8	N	N	N
34	AKBAR	54	M	4	N	Y	N	N	26.3	164	120	N	N	88	21	1.1	147	3.9	N	N	N
35	SUDHAKAR	51	M	4	N	N	N	N	28.8	168	108	N	N	69	26	0.8	142	3.9	L	C	N
36	NAGAMMAL	55	F	2	N	N	N	N	20.2	182	102	N	N	94	34	1	146	3.6	N	N	N

S. NO	NAME	U.VOLUME (ml)	U.PROTEIN (mg/24 hrs)	U.CREATININE (mg/24hrs)	U.SODIUM (mg/24hrs)	U.POTASSIUM (mg/24 hrs)
19	SAROJA	1200	55	1312	287	77
20	SARIKA	1350	57	1036	166	60
21	SARASA	950	34	1211	240	29
22	AMBIKA	1350	39	1206	187	33
23	ALAGAMMA	1600	75	1097	297	31
24	MUTHU	1450	47	1464	206	53
25	SELVAM	1450	40	1627	289	76
26	MOORTHY	1450	55	1547	258	27

27	AYISHA	1650	57	1259	219	60
28	BASHEERA	1350	42	1162	248	30
29	KAMATCHI	2200	48	957	123	55
30	RAJA	2250	79	1502	177	48
31	GOVINDASAMY	1350	46	1504	332	32
32	KANNAN	1250	72	1672	273	26
33	KAMALA	1050	63	1050	243	25
34	AKBAR	1750	84	1694	321	66
35	SUDHAKAR	1250	14	1060	253	27
36	NAGAMMAL	1400	49	1056	335	26

S.NO	NAME	AGE	SEX	SYMPTOM	P/H	SMOKING	ALCOHOL	F/H	B.M.I Kg/m²	S.B.P mm/Hg	D.B.P mm/Hg	FUNDUS	S.E	B.GLUCOSE	B.UREA	S.CREATININE	S.SODIUM	S.POTASSIUM	E.C.G	C.X.R	USG.ABD
37	VASANTHA	60	F	3	N	N	N	N	28.9	158	94	N	N	73	32	1.1	143	3.8	N	N	N
38	SEKAR	60	M	2	N	Y	N	Y	26.5	172	114	N	N	78	34	0.9	146	3.7	N	N	N
39	PUSHPA	59	F	1	N	N	N	N	21.6	168	108	N	N	105	26	0.8	145	4	N	N	N
40	CHANDRAN	55	M	2	N	N	N	N	20.2	172	112	N	N	74	32	1	139	4.3	N	N	N
41	AMEERA BEE	58	F	2	N	N	N	N	28.1	210	120	I	N	78	34	0.8	145	3.9	N	N	N

42	DURAI	54	M	2	N	N	N	N	25.7	190	114	N	N	98	32	1	144	4	N	N	N
43	THANMANI	54	F	4	N	N	N	Y	20.4	170	110	N	N	80	24	1	139	3.9	N	N	N
44	SOUNDARAM	56	F	2	N	N	N	N	20.2	192	106	N	N	97	26	0.6	142	4.3	N	N	N
45	SUDHAKAR	44	M	1	N	N	N	N	24.9	166	106	N	N	90	26	0.6	143	3.7	N	N	N
46	DHANABACKIYAM	50	F	3	N	N	N	N	29.7	186	108	N	N	93	24	0.8	146	3.7	N	N	N
47	MANI	60	M	2	N	Y	N	N	25.8	156	108	N	N	80	36	1.2	144	3.8	L	C	N
48	KUMAR	45	M	1	N	N	N	N	18.8	166	104	N	N	70	26	0.6	142	4.1	N	N	N
49	CHANDRAN	59	M	2	D	N	N	N	29.9	190	116	N	N	128	46	0.9	144	3.7	L	N	N
50	MANI	60	M	2	N	Y	Y	N	27.6	178	108	I	N	91	29	0.9	145	3.6	L	N	N
51	CHITTIBABU	52	M	3	N	N	N	Y	22.6	188	112	N	N	91	34	0.9	145	3.8	N	N	N
52	DURAIRAJ	53	M	5	N	Y	Y	N	29.1	210	120	N	N	63	42	0.9	146	3.9	N	N	N
53	MOIDEEN	50	M	2	N	Y	N	N	25.8	194	118	N	N	100	23	0.7	137	3.8	N	N	N
54	GOPAL	58	M	3	N	Y	Y	N	29.6	208	120	N	N	74	42	0.8	147	4	L	C	N

S.NO	NAME	U.VOLUME(ml)	U.PROTEIN (mg/24 hrs)	U.CREATININE (mg/24hrs)	U.SODIUM (mg/24hrs)	U.POTASSIUM (mg/24 hrs)
37	VASANTHA	1700	71	1103	316	24
38	SEKAR	1650	63	1752	191	74
39	PUSHPA	1200	54	1319	328	27

40	CHANDRAN	1250	71	1792	342	38														
41	AMEERA BEE	1500	48	1229	338	66														
42	DURAI	1650	72	1712	268	24														
43	THANMANI	1350	74	1195	286	24														
44	SOUNDARAM	2000	53	1202	172	45														
45	SUDHAKAR	1150	44	1283	276	55														
46	DHANABACKIYAM	1150	66	970	283	34														
47	MANI	1850	59	1237	172	63														
48	KUMAR	1400	69	1580	112	72														
49	CHANDRAN	1550	42	1304	310	34														
50	MANI	2250	26	1222	334	24														
51	CHITTIBABU	1250	101	1441	308	25														
52	DURAIRAJ	1400	66	1636	126	62														
53	MOIDEEN	1450	56	1973	217	35														
54	GOPAL	1750	60	1579	339	68														

S.NO	NAME	AGE	SEX	SYMPTO	P/H	SMOKIN	ALCOHO	F/H	B.M.I Kg/m²	S.B.P mm/Hg	D.B.P mm/Hg	FUNDUS	S.E	B.GLUCO	B.UREA	S.CREATINI NF	S.SODIU	S.POTASSIU M	E.C.G	C.X.R	USG.ABD
55	SANTHA	60	F	2	N	N	N	N	27.5	182	110	N	N	92	36	1.1	140	3.9	N	N	N

56	JOTHI	55	F	3	N	N	N	N	21.1	202	108	N	N	78	36	1	140	4	N	N	N
57	MANI	53	M	1	N	Y	N	B	27.5	176	102	I	N	124	44	0.9	140	4	N	N	N
58	DEVI	46	F	3	N	N	N	N	19.9	150	96	N	N	84	20	0.9	138	3.8	N	N	N
59	GOPAL	50	M	1	N	Y	Y	N	23.1	148	98	N	N	68	22	0.8	143	3.9	N	N	N
60	SUSEELA	50	F	2	N	N	N	N	27.3	146	92	N	N	100	20	0.9	139	3.9	N	N	N
61	VIJAYA	51	F	1	N	N	N	N	25.7	158	110	N	N	76	27	0.6	138	3.9	N	N	N
62	SAROJA	47	F	2	N	N	N	N	19.1	146	92	N	N	92	24	0.7	137	4	N	N	N
63	PERUMAL	53	M	1	N	N	N	Y	27.1	146	96	N	N	105	24	0.8	142	3.9	N	N	N
64	ISMAIL	44	M	2	N	N	N	N	21.9	140	94	N	N	93	22	0.7	137	3.6	N	N	N
65	SHANTHI	55	F	2	N	N	N	N	19.7	148	94	N	N	76	24	0.9	136	3.6	N	N	N
66	KRISHNAN	53	M	4	N	Y	N	Y	28.8	156	98	N	N	62	26	0.7	145	4	N	N	N
67	DHANRAJ	59	M	1	N	Y	N	N	21.1	146	96	N	N	77	28	0.7	138	4	N	N	N
68	MANOHAR	55	M	1	N	Y	Y	N	21.9	146	96	N	N	85	34	0.8	142	3.9	N	N	N
69	RAJENDRAN	43	M	1	D	Y	N	N	22.9	148	96	I	N	121	54	1	137	3.7	N	N	N
70	KAMALA	57	F	4	N	N	N	N	20.9	156	98	N	N	73	32	0.8	142	3.7	N	N	N
71	PALANI	45	M	0	N	N	N	N	18.9	90	70	N	N	115	22	0.8	141	4.4	N	N	N
72	SELVAM	46	M	0	N	N	N	N	23.7	98	69	N	N	64	22	0.8	144	4.2	N	N	N

S.NO	NAME	U.VOLUME (ml)	U.PROTEIN (mg/24 hrs)	U.CREATININE (mg/24hrs)	U.SODIUM (mg/24hrs)	U.POTASSIUM (mg/24 hrs)
55	SANTHA	2000	63	1136	332	63
56	JOTHI	1250	55	1016	313	41
57	MANI	1250	78	1641	120	36
58	DEVI	1200	38	1258	204	38
59	GOPAL	1400	44	1604	132	35
60	SUSEELA	1400	50	1176	229	34
61	VIJAYA	1350	92	927	226	24
62	SAROJA	1450	35	1160	138	25
63	PERUMAL	1200	53	1388	226	22
64	ISMAIL	1450	45	1711	132	61
65	SHANTHI	1500	64	1120	136	84
66	KRISHNAN	1350	44	2003	326	55
67	DHANRAJ	1050	80	1585	135	25
68	MANOHAR	1300	55	1452	179	37
69	RAJENDRAN	1500	57	1782	189	86
70	KAMALA	1350	39	1246	285	54
71	PALANI	1350	65	1594	135	69
72	SELVAM	1550	59	1269	247	51

S.NO	NAME	AGE	SEX	SYMPTOM	P/H	SMOKING	ALCOHOL	F/H	B.M.I Kg/m^2	S.B.P mm/Hg	D.B.P mm/Hg	FUNDUS	S.ш	B.GLUCOSE	B.UREA	S.CREATININE	S.SODIUM	S.POTASSIUM	E.C.G	C.X.R	USG.ABD
73	KANDASAMY	59	M	0	N	Y	N	N	17.9	101	70	N	N	82	56	0.9	136	4.3	N	N	N
74	KALIMA	49	F	0	N	N	N	N	24.4	102	72	N	N	89	36	0.9	137	4	N	N	N
75	MALARKODI	52	F	0	N	N	N	Y	22.6	102	72	N	N	69	33	0.9	139	3.8	N	N	N
76	MANGALAM	54	F	0	N	N	N	N	20.3	102	72	N	N	75	23	0.8	140	3.7	N	N	N
77	ABDUL	49	M	0	N	Y	Y	N	23.9	109	69	N	N	103	32	1	140	3.8	N	N	N
78	KAVERI	48	F	0	N	N	N	N	26.4	110	69	N	N	66	34	0.8	140	3.7	N	N	N
79	USHA	53	F	0	N	N	N	D	22.2	110	70	N	N	96	20	0.8	138	4	N	N	N
80	UMA	54	F	0	N	N	N	N	19.1	110	70	N	N	78	24	0.9	138	3.9	N	N	N
81	PAKKIRI	48	M	0	D	Y	N	D	17.7	110	70	N	N	132	60	1.1	135	3.8	N	N	N
82	MURUGAN	48	M	0	N	Y	Y	N	21.2	110	80	N	N	66	32	0.6	138	4	N	N	N
83	MANI	48	M	0	D	Y	N	N	23	110	70	N	N	133	54	0.6	139	3.9	N	N	N
84	MADAN	56	M	0	N	N	N	N	20.5	110	70	N	N	96	22	0.8	137	4.1	N	N	N
85	KRISHNAN	56	M	0	N	Y	Y	N	22.9	110	70	N	N	108	21	0.8	142	4.1	N	N	N
86	MOORTHY	58	M	0	N	Y	N	N	19.3	110	70	N	N	110	24	0.9	136	4.1	N	N	N

87	AVITTAN	60	M	0	N	Y	N	N	22	110	70	N	N	95	22	1.1	136	3.9	N	N	N
88	ANANDHAN	46	M	0	N	N	N	N	23.2	99	69	N	N	90	22	0.9	138	3.9	N	N	N
89	AMBIKA	54	F	0	N	N	N	N	20.9	100	70	N	N	68	41	0.9	138	4.2	N	N	N
90	MUNUSAMY	50	M	0	N	Y	Y	Y	22.6	100	60	N	N	64	20	0.8	145	3.8	N	N	N

S.NO	NAME	U.VOLUME (ml)	U.PROTEIN (mg/24 hrs)	U.CREATININE (mg/24hrs)	U.SODIUM (mg/24hrs)	U.POTASSIUM (mg/24 hrs)
73	KANDASAMY	1600	53	1211	128	68
74	KALIMA	1300	47	1383	158	42
75	MALARKODI	1250	46	1087	238	22
76	MANGALAM	1450	59	1391	256	71
77	ABDUL	1300	28	1119	254	79
78	KAVERI	1400	82	927	262	41
79	USHA	1600	47	850	208	48
80	UMA	1200	53	1178	140	61
81	PAKKIRI	1600	47	1269	234	60
82	MURUGAN	1350	70	1483	198	30
83	MANI	1650	46	1434	110	82
84	MADAN	1450	44	1497	164	60
85	KRISHNAN	1350	49	1166	228	54

86	MOORTHY	1300	76	1513	145	37
87	AVITTAN	1450	57	1558	206	22
88	ANANDHAN	1400	59	1648	149	20
89	AMBIKA	1350	56	1254	180	47
90	MUNUSAMY	1650	74	1888	237	55

S.NO	NAME	AGE	SEX	SYMPTOM	P/H	SMOKING	ALCOHOL	F/H	B.M.I Kg/m^2	S.B.P mm/Hg	D.B.P mm/Hg	FUNDUS	S.ш	B.GLUCOSE	B.UREA	S.CREATININ	S.SODIUM	S.POTASSIUM	E.C.G	C.X.R	USG.ABD
91	KAYALVILI	56	F	0	N	N	N	N	22.3	110	70	N	N	92	22	0.9	138	4.1	N	N	N
92	PAL VANNAN	58	M	0	N	Y	N	N	21.2	110	80	N	N	69	25	0.9	139	4.1	N	N	N
93	FATHIMA	47	F	0	N	N	N	N	20	110	70	N	N	67	32	1	142	3.8	N	N	N
94	UMA DEVI	54	F	0	N	N	N	N	20.2	100	70	N	N	101	43	1	139	4.1	N	N	N
95	SRINIVASAN	60	M	0	N	Y	N	N	19.3	100	72	N	N	84	24	1.1	136	4.2	N	N	N
96	SIVA	57	M	0	N	N	N	N	23.3	112	72	N	N	90	28	0.7	143	4	N	N	N
97	RAVI	47	M	0	N	Y	Y	N	21.3	112	82	N	N	76	26	0.9	138	4	N	N	N
98	RAZA BEE	55	F	0	N	N	N	N	20.5	114	74	N	N	110	34	0.9	143	4.1	N	N	N

99	MUNIAN	48	M	0	N	Y	N	N	22.7	116	76	N	N	97	32	1	138	3.9	N	N	N
100	THOMAS	43	M	0	N	Y	N	N	17.3	120	80	N	N	91	24	0.8	136	4.5	N	N	N

S.NO	NAME	U.VOLUME (ml)	U.PROTEIN (mg/24 hrs)	U.CREATININE (mg/24hrs)	U.SODIUM (mg/24hrs)	U.POTASSIUM (mg/24 hrs)
91	KAYALVILI	1400	50	1477	214	55
92	PAL VANNAN	1600	71	1073	186	51
93	FATHIMA	1050	70	1602	153	39
94	UMA DEVI	1150	76	1289	155	68
95	SRINIVASAN	1350	78	1968	151	29
96	SIVA	1600	70	942	222	35
97	RAVI	1000	76	1326	202	58
98	RAZA BEE	1350	49	1583	173	80

99	MUNIAN	1250	54	1099	222	60
100	THOMAS	1250	61	1415	88	61

ABBREVIATION USED IN MASTER CHART

0 - No symptom

1 – Giddiness

2 – Headache

3 - Chest pain

4 - Palpitation

5 - Dyspnoea

S.B.P – Systolic blood pressure

D.B.P –Diastolic blood pressure

Y-yes N-normal

M-male F-female

S.E –systemic examination

I - Grade I hypertension retinopathy

L - Left ventricular hypertrophy

H - LAHB

C - Cardiomegaly

D- Diabetes

B- Both Diabetes and Hypertension

F/H - Family history

P/H - Past history